PROCALCITONIN AND MALONDIALDEHYDE AS MARKERS OF INFLAMMATION IN HEMODIALYSIS PATIENTS

HEMODİYALİZ HASTALARINDA YENİ BİR İNFLAMASYON GÖSTERGESİ OLARAK PROKALSİTONİN VE MALONDİALDEHİD

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This study was Dr. Nigar Sekercioglu's nephrology thesis.

Öz

Amaç: Prokalsitonin enfeksiyon markırı olarak bilinir. Malondialdehid oksidatif stres markırı kabul edilir. Periton diyaliz hastalarında iki markır da yüksek olarak tesbit edilmiştir. Bu çalışmada prokalsitonin, malondialdehid, C reaktif protein, albümin, fıbrinojen, lokosit, lenfosit ve sedimentasyon gibi inflamasyon markırları hemodiyaliz hastalarında, orta derecede böbrek yetersizliği olan hasta grubunda ve sağlıklı kontrol grubunda belirlendi. Gereç ve Yöntem: 35 Hemodiyaliz hastası, 30 kreatinin klirensi 39.9±9.9ml/dk olan hasta ve 20 kişiden oluşan kontrol grubunda plazma prokalsitonin, malondialdehid, C reaktif protein, albümin, fıbrinojen, lokosit, lenfosit ve sedimentasyon düzeyleri belirlendi. Prokalsıtoninin ve malondialdehidin diğer markırlarla olan korelasyonu değerlendirildi. Bulgular: Hemodiyaliz hastalarında prokalsitonin düzeyi anlamlı olarak yüksekti ($p<0.01$) ve C reaktif protein düzeyi ile koreleydi ($r:0.89$). Tartışma: Hemodiyaliz hastalarında yüksek prokalsitonin düzeyi minimal endotoksin kontaminasyonundan kaynaklanmış olabilir. Çalışmamızda, inflamasyon markırı olarak bilinen yüksek C reaktif protein düzeyi ile prokalsitonin düzeyi arasında korelasyon tesbit ettik. Prokalsitonin inflamasyon markırı olabilir.

Anahtar Kelimeler

Kronik Böbrek Hastalığı; Malondialdehid; Prokalsitonin.

Abstract

Aim: Procalcitonin (PCT) is known as an infection marker. Malondialdehyde (MDA) is considered as a marker of oxidative stress. Both have been found to be elevated in peritoneal dialysis patients. In this study, we measured procalcitonin, malondialdehyde, and traditional inflammation markers, including albumin, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), fibrinogen, lymphocyte counts, and white blood cell counts (WBC) in hemodialysis (HD) patients, patients with moderate chronic kidney disease (CKD), and healthy subjects. Material and Method: We measured PCT, MDA, albumin, CRP, ESR, fibrinogen, lymphocyte counts, and WBC in 35 maintenance HD patients, 30 patients with moderate chronic kidney disease and a control group with normal kidney functions. The correlation of PCT and MDA with traditional inflammation markers (albumin, CRP, ESR, fibrinogen, lymphocyte counts, and WBC) was also evaluated. Results: PCT levels were significantly higher and were correlated with CRP levels in the HD group ($r=0.89$). MDA was significantly higher in HD patients and non-dialysis CKD patients as compared to healthy controls. Discussion: Higher PCT levels in HD patients might be caused by minimal endotoxin contamination. Elevated CRP levels, known as a marker of inflammation, were correlated with elevated PCT levels. PCT may also be a marker of inflammation.

Keywords

Chronic Kidney Disease; Malondialdehyde; Procalcitonin.

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Introduction

Chronic kidney disease (CKD) is global health problem affecting about 10% of the world population [1]. The condition is related to high mortality, morbidity, health care resource use, and costs [3-4]. In patients with chronic kidney disease, the most common cause of death is cardiovascular diseases and followed by infections [5-7]. Therefore, it is important to establish reliable markers of bacterial infections to optimize management of infections in the CKD patient population.

The prevalence of malnutrition, inflammation, and atherosclerosis—also called MIA syndrome-—is high in patients with CKD and signifies oxidative stress and cardiovascular risk [2, 3]. In addition to traditional cardiovascular risk factors, non-traditional cardiovascular risk factors also need to be addressed in the management of CKD patients to improve quality and quantity of life [8,9].

Indicators of disease activity in CKD patients have been an area of interest for many researchers. Classical markers of infections and inflammation include albumin, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and white blood cell counts (WBC). CRP is a cytokine mediated acute phase reactant. The marker guides clinicians to follow disease activity and prognosis [10]. However, CRP has not proven to be significant for mortality in hemodialysis (HD) patients [11].

Procalcitonin (PCT) is a precursor of calcitonin which is secreted by the thyroid C cells, also called parafollicular cells [7, 12-14]. PCT is secreted by the liver in bacterial infections and exceed the normal limit of 0.1 ng/ml in the normal population [9, 12-14]. Malondialdehyde is a marker of oxidative stress and has been shown to be elevated in dialysis patients [8,15].

Our objective was to assess serum levels of MDA (a marker of oxidative stress) and of PCT (a marker of bacterial infections and systematic inflammation) in patients who were on HD three times per week (group A); in patients with an estimated glomerular filtration rate (eGFR) of less than 60 ml/min/1.73 m2 (group B); and in healthy subjects with GFR of more than 90 ml/min/1.73 m2 (group C). We measured the difference in PCT, MDA, albumin, CRP, ESR, fibrinogen, lymphocyte counts, and WBC between the groups. We also explored the correlation between PCT and MDA with albumin, CRP, ESR, fibrinogen, lymphocyte counts, and WBC.

Material and Method

Patients

This study was based on the nephrology thesis of Dr. Nigar Sekercioglu. This is a cross-sectional study of sixty-five adult patients with stages 4 through 5D CKD and twenty healthy controls. HD patients were recruited from the University of Istanbul Cerrahpasa Faculty of Medicine Hospital Hemodialysis Unit. Patients with CKD were recruited from the University of Istanbul Cerrahpasa Faculty of Medicine Hospital outpatient nephrology clinics.

Kidney dysfunction was defined by eGFR calculated with the MDRD equation (186*[serum creatinine (mg/dl)]-1.154*[age (years)]-0.203*[0.742 (if female)]) using a specific calculator designed for eGFR estimation with the MDRD equation [16].

Inclusion criteria included (1) being an adult patient (18 years), (2) being on HD for three times per week for the group A, (3) having some degree of kidney dysfunction with a GFR cut-off level of 60 L/min/1.73m2 for group B, and (4) agreeing to participate in the study. Exclusion criteria included those with: (1) tuberculosis, (2) known bacterial infections, (3) diabetes mellitus, (4) active vasculitis, or (5) inflammatory bowel diseases (Crohn's or ulcerative colitis).

This study was approved by the University of Istanbul Cerrahpasa Faculty of Medicine Research Ethics Authority. An informed consent was obtained from each participant.

One fasting blood sample was collected from all participants and sent to the Central Research Laboratory for analysis. The blood samples were centrifuged and stored at -60 C0 and were analyzed immediately after being thawed. In HD patients, we collected blood samples before starting dialysis. Samples were collected on Monday from patients on a Monday-Wednesday-Friday schedule and on Tuesday from those on a Tuesday-Thursday-Saturday schedule.

PCT was measured using DiaSorin assay (DiaSorin, Saluggia, Italy). The normal cut-off value for PCT was <0.5ng/ml. MDA was measured using the technique suggested by Buege and August with thiobarbituric acid reactive substances [17].

Albumin was measured using the Olympus AU 800 Analyzer (Olympus, Japan). CRP was measured using a high sensitivity immunonephelometric technique (Dade Behring, Marburg, Germany). In healthy subjects, the normal limits of CRP range from 0 to 5 mg/L.

Fibrinogen was measured using a Dade Behring Dimension Analyzer (Dade Behring, Germany). ESR was measured using an Eriline AR Linear (Barcelona, Spain) device. Albumin was measured using an Olympus AU 800® Analyzer (Japan). WBC and lymphocyte counts were measured using an HmX Hematology Analyzer (Beckman Coulter, USA).

Statistical analysis

The analysis plan included descriptive and inferential statistics. Counts and percents were used to summarize categorical variables, while means and standard deviations (SD) were used for continuous variables. We employed the Pearson's Chi Square test to examine the distribution of patients' characteristics for categorical variables. We employed two-sample t tests to compare mean differences between the groups for continuous variables.

MDA and PCT were tested for association with various covariates using the Spearman's coefficient of correlation (r in the tables). Two-sided tests were employed with a significance level of 0.05. All data analyses were performed using IBM SPSS Statistics 20, Release Version 10.0 (SPSS Inc., Chicago, IL; www.spss.com).

Results

Our study cohort consisted of eighty-five subjects. In this cohort, the most common cause of end stage renal disease was chronic glomerulonephritis, and hypertension was the most common cause of underlying CKD in non-dialysis CKD patients. Healthy controls, patients with moderate chronic kidney disease and HD patients had mean eGFR of 111 (SD=11.3), 39.9 (SD=9.9), and 9.8 (SD=16) ml/min/1.73m2, respectively.

The group A consisted of 35 HD patients (25 males and 10

Table 1. Baseline characteristics of the participants

	Group A (n=35)	Group B (n=30)	Group C (n=20)
Age (years); mean (SD)	47.5 (15.3)	49.7 (19.6)	41.2 (16.3)
Female gender; n (%)	10 (28%)	12 (40%)	6 (30%)
Dialysis vintage(months); mean (SD)	21 (12)	Not applicable	Not applicable
eGFR (ml/min/1.73m2)	9.8 (1.6)	39.9 (9.9)	111 (11.3)
Polycystic kidney disease; n (%)	5	1	Not applicable
Minimal change disease; n (%)	0	1	Not applicable
Membranous glomerulonephritis; n (%)	0	5	Not applicable
Membranoproliferative glomerulonephritis; n (%)	0	4	Not applicable
Focal segmental glomerulosclerosis; n (%)	0	1	Not applicable
IgA nephropathy; n (%)	0	1	Not applicable
Rapidly progressive glomerulonephritis; n (%)	1	0	Not applicable
Hypertension; n (%)	7	17	Not applicable
Unknown etiology; n (%)	4	0	Not applicable
Chronic glomerulonephritis; n (%)	18	0	Not applicable

Note: Group A indicates hemodialysis patients; group B indicates non-dialysis CKD patients and group C indicates healthy controls. Abbreviations: eGFR: Estimated glomerular filtration rate.

females). The mean age of these patients was 47.5 years (SD= 15.3) (Table 1). Of these 35 patients, 18 had chronic glomerulonephritis, seven had hypertension, five had polycystic kidney disease, four had unknown etiology, and one patient had rapidly progressive glomerulonephritis (Table 1). The mean dialysis vintage was 21.9 months (SD=12.6).

We included 30 patients in the group B (18 males and 10 females). The mean age in the group B was 49 years (SD=18.6). A total of 17 patients had hypertension in the group B. Five patients had membranous glomerulonephritis while 4 patients had membranoproliferative glomerulonephritis. The mean eGFR of non-dialysis CKD patients was 39.9 ml/min/1.73m2.

We included 20 healthy subjects in the group C (14 males and 8 females), for which the mean age was 41.2 years. GFR was above 90/ml/min/1.73m2 (mean was 111 ml/min/1.73 m2) in the group C.

MDA was significantly higher in the group A as compared to the group B and group C. Also, MDA was significantly higher in the group B as compared to the group C (Table 2).

PCT was significantly higher in the HD patients than patients in groups B and C. However, the difference in PCT did not reach a statistically significant level between groups B and C. Fibrinogen, WBC, and CRP did not indicate significantly different results between the groups. Lymphocyte count was significantly lower in groups A and B when compared to the group C. Nevertheless, there was no significant difference in lymphocyte count between groups A and B. Albumin was significantly lower in HD patients as compared to healthy controls. However, the difference for albumin was not significant between groups B and C. ESR was significantly higher in HD patients as compared to groups B and C. However, there was no significant difference in ESR between groups B and C (Table 2).

Table 2. Laboratory data across groups

	Group A (N=35)	Group B (n=30)	Group C (n=20)	P-value
MDA (nmol/ml)	8.04 (1.05)	7.16 (1.50)	6.24 (1.46)	A vs B= 0.02 A vs C= 0.001 B vs C= 0.04
PCT	0.81 (1.29)	0.14 (0.08)	0.11 (0.01)	A vs B= 0.001 A vs C= 0.01 B vs C= 0.98
WBC	6757 (2165)	7886 (2151)	8030 (1766)	A vs B= 0.07 A vs C= 0.07 B vs C= 0.96
Lymphocyte	2142 (550)	2139 (637)	2679 (612)	A vs B= 1 A vs C= 0.05 B vs C= 0.05
Albumin (g/dl)	3.09 (0.53)	3.38 (0.69)	3.58 (0.60)	A vs B= 0.13 A vs C= 0.01 B vs C= 0.48
Sedimentation	58 (26)	35 (21)	32 (26)	A vs B= 0.001 A vs C= 0.001 B vs C= 0.92
Fibrinogen (mg/dl)	680 (869)	407 (159)	321 (133)	A vs B= 0.14 A vs C= 0.07 B vs C= 0.86
CRP (mg/L)	13 (30)	6.14 (7.20)	7.7 (15)	A vs B= 0.38 A vs C= 0.64 B vs C= 0.96

Note: Group A indicates hemodialysis patients; group B indicates non-dialysis CKD patients and group C indicates healthy controls. Abbreviations: CRP: C-recative protein, MDA: Malondialdhyde, PCT: Procalcitonin, SD: standard deviations, WBC: white blood cell counts

Table 3. Correlation analysis in the hemodialysis group

	PCT (r; p value)	MDA (r; p value)
MDA	0.32 (0.059)	-
Albumin	-0.17 (0.31)	-0.30 (0.07)
CRP	0.89 (0.0001)	0.25 (0.13)
Fibrinogen	0.006 (0.97)	-0.08 (0.62)
WBC	-0.079 (0.65)	0.30 (0.07)
Lymphocyte	-0.073 (0.67)	0.0001 (1)
Sedimentation	0.22 (0.18)	0.008 (0.96)
Age	0.04 (0.78)	-0.06 (0.71)
BUN	-0.13 (0.43)	0.07 (0.67)
Creatinine	-0.05 (0.74)	-0.01 (0.94)

Abbreviations: CRP: C-recative protein, MDA: Malondialdhyde, PCT: Procalcitonin, SD: standard deviations, WBC: white blood cell counts

In the group A, only CRP was positively correlated with PCT (r=0.89, p=0.0001) (Table 3). In the group B, fibrinogen and creatinine were positively correlated with PCT (r=0.36, p=0.04 for fibrinogen and r=0.38, p=0.03 for creatinine) (Table 4). In the group C, BUN was negatively correlated with PCT (r=-049, p=0.02) (Table 5). Other covariates did not reach a significant level (Table 5).

In the group A, none of the variables indicated significant correlation with MDA (Table 3). In the group B, none of the variables indicated significant correlation with MDA (Table 4). In the group C, CRP and ESR were positively correlated with MDA (r=0.75, p=0.0001 for CRP and r=0.49, p=0.02 for ESR). Other covariates did not reach a significant level (Table 5).

Discussion

Our aim was to explore the difference in PCT, MDA, albumin, CRP, ESR, fibrinogen, lymphocyte counts, and WBC between pa-

Table 4. Correlation analysis in the non-dialysis CKD patients

	PCT; r (p value)	MDA; r (p value)
MDA	-0.01 (0.95)	-
Albumin	-0.11 (0.56)	-0.05 (0.79)
CRP	-0.08 (0.63)	0.05 (0.76)
Fibrinogen	0.36 (0.04)	0.12 (0.52)
WBC	-0.29 (0.11)	0.04 (0.79)
Lymphocyte	-0.02 (0.90)	0.007 (0.96)
Sedimentation	0.07 (0.68)	-0.08 (0.67)
Age	-0.17 (0.36)	0.22 (0.23)
BUN	0.28 (0.13)	-0.15 (0.40)
Creatinine	0.38 (0.03)	-0.20 (0.28)

Abbreviations: CKD: Chronic kidney disease; CRP: C-recative protein, MDA: Malondialdhyde, PCT: Procalcitonin, SD: standard deviations, WBC: white blood cell counts

Table 5. Correlation analysis in healthy controls

	PCT (r; p value)	MDA (r; p value)
MDA	0.10 (0.66)	-
Albumin	-0.11 (0.62)	-0.22 (0.35)
CRP	-0.17 (0.46)	0.75 (0.0001)
Fibrinogen	-0.34 (0.14)	-0.13 (0.58)
WBC	0.12 (0.59)	0.24 (0.29)
Lymphocyte	-0.25 (0.28)	-0.14 (0.54)
Sedimentation	-0.24 (0.30)	0.49 (0.02)
Age	-0.32 (0.16)	0.28 (0.21)
BUN	-0.49 (0.02)	0.08 (0.71)
Creatinine	-0.23 (0.33)	0.15 (0.52)

Abbreviations: CRP: C-recative protein, MDA: Malondialdhyde, PCT: Procalcitonin, SD: standard deviations, WBC: white blood cell counts

tients with different levels of kidney dysfunction and healthy controls. We also explored the correlation between PCT and MDA with other inflammation markers.

HD patients showed significantly higher PCT, MDA, and ESR levels as compared to non-dialysis CKD patients and healthy controls. Fibrinogen, WBC, and CRP did not indicate significantly different results between the groups, while albumin was significantly lower in HD patients as compared to healthy controls. With regards to correlations between the markers, CRP was positively correlated with PCT in HD patients.

Our results indicated that PCT was significantly higher in HD patients. Nevertheless, PCT was not significantly higher in non-dialysis CKD patients as compared to healthy controls in our study. Results from previous reports support our findings. Visardis et al. investigated the correlation between PCT and traditional inflammation markers. Results indicated that 38% of HD patients had elevated PCT levels [18]. Herge et al. found a negative correlation between GFR and PCT level. The authors attributed the elevated levels to increased synthesis of PCT from peripheral blood mononuclear cells and reduced clearance of PCT by the kidneys [19].

Opatrna et al. showed that PCT was significantly higher in peritoneal dialysis (PD) patients as compared to healthy controls [20]. In line with these findings, another study showed normal levels of PCT in HD patients and elevated levels of PCT in PD patients without bacterial infection [7].

However, Sitter et al. failed to show a significant correlation between PCT and eGFR [21]. Thus, the authors concluded that PCT as a marker of infection had acceptable diagnostic properties (sensitivity and specificity) for systematic bacterial infections in CKD patients [21].

In our study, the correlation of CRP with PCT was significant (p=0.0001) with a coefficient of r=0.89. This is supported by previous reports. CRP was positively correlated with PCT in HD patients (r=0.50) [20] and PD patients (r=0.59) [21]. Since CKD patients show elevated CRP levels, the marker has been associated with more false positive results in CKD patients [18, 21].

CRP has been found to be relatively stable after dialysis. Dahaba et al. found significantly lower PCT values after dialysis while CRP levels remained the same [22]. Nondialyzable inflammation markers are useful tools for dialysis patients. However, it is important to connect these surrogate markers to patient-important outcomes, such as mortality. Owen et al. failed to show a significant relationship between CRP and mortality [11].

Our results showed lower levels of serum albumin in HD patients as compared to non-dialysis CKD patients and health controls. Tombul et al. studied 60 dialysis patients (30 HD and 30 PD) to test the difference in inflammation markers (albumin, CRP, ESR and fibrinogen). The results indicated that albumin was lower in PD patients [23].

MDA was significantly higher in HD patients and non-dialysis CKD patients as compared to healthy controls. This is congruent with previous reports. Miller et al. reported that MDA was elevated in dialysis patients [24]. Furthermore, a study investigated the differences in ESR, CRP, and MDA in patients with CKD and health controls [25]. Those with CKD had elevated serum levels of CRP and MDA [25].

Ours was a cross-sectional study of CKD patients and health controls. We pre-defined our research question and objectives. We employed statistical methods to explore: (1) the differences between the groups, and (2) the correlation between inflammation markers using the frequentist approach. However, there are several limitations that need to be addressed. First, this is a cross-sectional study and may be used only for hypothesis-generating. Secondly, we did not include PD patients and kidney transplant recepients. Thus, results may not be applicable for patients who were on different renal replacement therapies.

Overall, we reported that dialysis and non-dialysis CKD patients showed elevated PCT and MDA levels. Albumin was lower in dialysis patients as compared to healthy controls. Our findings support the presence of chronic inflammation in CKD patients. The remaining important question is the relationship of these markers with important patient outcomes, such as renal and patient survival.

Competing interests

The authors declare that they have no competing interests.

References

1. Coresh J, Selvin E, Stevens LA, Manzi J, Kusek JW, Eggers P, et al. Prevalence of chronic kidney disease in the United States. JAMA 2007;298(17):2038-47.
2. Chowdhury R, Peel NM, Krosch M, Hubbard RE. Frailty and chronic kidney disease: A systematic review. Archives of gerontology and geriatrics 2016; 68:135-42.
3. Rao P, Reddy GC, Kanagasabapathy AS. Malnutrition-inflammation-atherosclerosis syndrome in Chronic Kidney disease. Indian Journal of Clinical Biochemistry 2008;23(3):209-17. 4. Foley RN, Murray AM, Li S, Herzog CA, McBean AM, Eggers PW, et al. Chronic kidney disease and the risk for cardiovascular disease, renal

replacement, and death in the United States Medicare population, 1998 to 1999. Journal of the American Society of Nephrology: JASN 2005;16(2):489-95.
5. Tonelli M, Wiebe N, Culleton B, House A, Rabbat C, Fok M, et al. Chronic kidney disease and mortality risk: a systematic review. Journal of the American Society of Nephrology: JASN. 2006;17(7):2034-47.
6. Hemmelgarn BR, Manns BJ, Lloyd A, James MT, Klarenbach S, Quinn RR, et al. Relation between kidney function, proteinuria, and adverse outcomes. JAMA 2010;303(5):423-9.
7. Steinbach G, Bolke E, Grunert A, Storck M, Orth K. Procalcitonin in patients with acute and chronic renal insufficiency. Wiener klinische Wochenschrift 2004;116 (24):849-53.
8. Cachofeiro V, Goicochea M, de Vinuesa SG, Oubina P, Lahera V, Luno J. Oxidative stress and inflammation, a link between chronic kidney disease and cardiovascular disease. Kidney international Supplement 2008;111:S4-9.
9. Kendrick J, Chonchol MB. Nontraditional risk factors for cardiovascular disease in patients with chronic kidney disease. Nature clinical practice Nephrology 2008;4(12):672-81.
10. Deodhar SD. C-reactive protein: the best laboratory indicator available for monitoring disease activity. Cleveland Clinic Journal of Medicine 1989;56(2):126-30.
11. Owen WF, Lowrie EG. C-reactive protein as an outcome predictor for maintenance hemodialysis patients. Kidney International 1998;54(2):627-36.
12. Assicot M, Gendrel D, Carsin H, Raymond J, Guilbaud J, Bohuon C. High serum procalcitonin concentrations in patients with sepsis and infection. Lancet (London, England) 1993;341(8844):515-8.
13. Bossink AW, Groeneveld AB, Thijs LG. Prediction of microbial infection and mortality in medical patients with fever: plasma procalcitonin, neutrophilic elastase-alpha1-antitrypsin, and lactoferrin compared with clinical variables. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America 1999;29(2):398-407.
14. Ouseph R, Ward RA. Ultrapure dialysate for home hemodialysis? Advances in Chronic Kidney Disease 2007;14(3):256-62.
15. Rasic S, Rebic D, Hasic S, Rasic I, Delic Sarac M. Influence of Malondialdehyde and Matrix Metalloproteinase-9 on Progression of Carotid Atherosclerosis in Chronic Renal Disease with Cardiometabolic Syndrome. Mediators of inflammation 2015; 2015:614357.
16. Levey AS, Coresh J, Balk E, Kausz AT, Levin A, Steffes MW, et al. National Kidney Foundation practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Annals of Internal Medicine 2003;139(2):137-47.
17. Buege JA, Aust SD. Microsomal lipid peroxidation. In: Fleisher S, Packer I, editors. Methods in Enzimology. New York: Academic Press; 1978. p.301-310.
18. Visvardis G, Griveas I, Fleva A, Giannakou A, Papadopoulou D, Mitsopoulos E, et al. Relevance of procalcitonin levels in comparison to other markers of inflammation in hemodialysis patients. Renal Failure 2005;27(4):429-34.
19. Herget-Rosenthal S, Klein T, Marggraf G, Hirsch T, Jakob HG, Philipp T, et al. Modulation and source of procalcitonin in reduced renal function and renal replacement therapy. Scandinavian Journal of Immunology 2005;61(2):180-6.
20. Opatrna S, Klaboch J, Opatrny K, Jr., Holubec L, Tomsu M, Sefrna F, et al. Procalcitonin levels in peritoneal dialysis patients. Peritoneal Dialysis international 2005;25(5):470-2.
21. Sitter T, Schmidt M, Schneider S, Schiffl H. Differential diagnosis of bacterial infection and inflammatory response in kidney diseases using procalcitonin. Journal of Nephrology 2002;15(3):297-301.
22. Dahaba AA, Rehak PH, List WF. Procalcitonin and C-reactive protein plasma concentrations in nonseptic uremic patients undergoing hemodialysis. Intensive Care Medicine 2003;29(4):579-83.
23. Tonbul HZ, Demir M, Altintepe L, Guney I, Yeter E, Turk S, et al. Malnutrition-inflammation-atherosclerosis (MIA) syndrome components in hemodialysis and peritoneal dialysis patients. Renal Failure 2006;28(4):287-94.
24. Miler M, Zamojska S, Fijalkowski P, Lobos M, Paradowski MT. [Assessment of malonyldialdehyde concentration as a product of lipid peroxidation and lipid metabolism in patients on chronic dialysis]. Polski Merkuriusz Lekarski : Organ Polskiego Towarzystwa Lekarskiego 2006;20(120):664-7. .
25. Ece A, Gurkan F, Kervancioglu M, Kocamaz H, Gunes A, Atamer Y, et al. Oxidative stress, inflammation and early cardiovascular damage in children with chronic renal failure. Pediatric nephrology (Berlin, Germany) 2006;21(4):545-52.

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